

\* \* \* \* \* STN Columbus \* \* \* \* \*

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=> file ca

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SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

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FILE COVERS 1907 - 8 Sep 2005 VOL 143 ISS 12

FILE LAST UPDATED: 8 Sep 2005 (20050908/ED)

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=> e 102:179661/an

E1	1	102:17966/AN
E2	1	102:179660/AN
E3	1 -->	102:179661/AN
E4	1	102:179662/AN
E5	1	102:179663/AN
E6	1	102:179664/AN
E7	1	102:179665/AN
E8	1	102:179666/AN
E9	1	102:179667/AN
E10	1	102:179668/AN
E11	1	102:179669/AN
E12	1	102:17967/AN

=> s e3

L1 1 "102:179661"/AN

=> d

L1 ANSWER 1 OF 1 CA COPYRIGHT 2005 ACS on STN

AN 102:179661 CA

TI Norepinephrine amplifies human chorionic gonadotropin-stimulated androgen biosynthesis by ovarian theca-interstitial cells

AU Dyer, Cheryl A.; Erickson, Gregory F.

CS Dep. Reprod. Med., Univ. California, San Diego, La Jolla, CA, 92093, USA

SO Endocrinology (1985), 116(4), 1645-52

CODEN: ENDOAO; ISSN: 0013-7227

DT Journal

LA English

=> e 97:157029/an

E1	1	97:157027/AN
E2	1	97:157028/AN
E3	1 -->	97:157029/AN
E4	1	97:15703/AN
E5	1	97:157030/AN
E6	1	97:157031/AN
E7	1	97:157032/AN
E8	1	97:157033/AN
E9	1	97:157034/AN
E10	1	97:157035/AN
E11	1	97:157036/AN
E12	1	97:157037/AN

=> s e3

L2 1 "97:157029"/AN

=> d

L2 ANSWER 1 OF 1 CA COPYRIGHT 2005 ACS on STN

AN 97:157029 CA

TI Catecholamine stimulation of androgen production by mouse interstitial cells in primary culture

AU Moger, William H.; Murphy, Paul R.; Casper, Robert F.

CS Dep. Physiol. Biophys., Dalhousie Univ., Halifax, NS, Can.

SO Journal of Andrology (1982), 3(4), 227-31

CODEN: JOAND3; ISSN: 0196-3635

DT Journal

LA English

=> d ab 11

L1 ANSWER 1 OF 1 CA COPYRIGHT 2005 ACS on STN

AB Ovarian theca-interstitial cells, when cultured in serum-free medium, secreted androgens in response to human chorionic gonadotropin (hCG) [9002-61-3] stimulation. This production was dependent on time (maximum production

attained after 96 h) and dose (half-maximal ED of hCG, 9 ng/mL). When the sympathomimetics norepinephrine [51-41-2], epinephrine [51-43-4], and isoproterenol [7683-59-2] were added to the medium, androgen production in response to hCG was enhanced by 100-300%. The ability of the catecholamines to stimulate androgen production was dependent on the continuous presence of hCG. Treatment with catecholamines alone did not induce theca-interstitial cells to produce androgens. Catecholamine stimulation of steroid hormone metabolism was selective for intermediates in the  $\Delta 4$ -pathway, with greatest increases occurring in the production of androstenedione [63-05-8] and testosterone [58-22-0]. The effect of the catecholamines on androgen production was dependent on both  $\beta 1$ - and  $\beta 2$ -adrenergic receptors. The acquisition of catecholamine responsiveness was specific to hCG; if theca-interstitial cells were induced to differentiate with either PGE2 or cholera toxin, then isoproterenol did not enhance androgen synthesis. The catecholamine-induced increases in androgen production were not due to a granulosa cell contribution of steroid. The interstitial cells are the only steroid-producing cells in the ovary that are directly innervated by norepinephrine-containing fibers of the sympathetic nervous system. This catecholamine-augmented androgen production provides a direct link between the autonomic nervous system and regulation of ovarian steroid synthesis.

=> d ab 12

L2 ANSWER 1 OF 1 CA COPYRIGHT 2005 ACS on STN  
AB The catecholamines 1-isoproterenol [2964-04-7], 1-epinephrine [51-43-4], and (-)-norepinephrine [51-41-2] stimulated androgen production by mouse interstitial cells in primary culture. The amount of androgen produced in response to maximum stimulation with these amines was less than that produced with maximum human chorionic gonadotropin (hCG) [9002-61-3] stimulation, but produced an additive effect when combined with a submaximal concentration of hCG.

The stimulatory effect of isoproterenol was blocked by the  $\beta$ -receptor antagonist propranolol. Isoproterenol did not stimulate androgen production by either freshly isolated mouse interstitial cells or whole decapsulated testes.

=> e 97:36514/an

E1	1	97:36512/AN
E2	1	97:36513/AN
E3	1 -->	97:36514/AN
E4	1	97:36515/AN
E5	1	97:36516/AN
E6	1	97:36517/AN
E7	1	97:36518/AN
E8	1	97:36519/AN
E9	1	97:3652/AN
E10	1	97:36520/AN
E11	1	97:36521/AN
E12	1	97:36522/AN

=> s e3

L3 1 "97:36514"/AN

=> d

L3 ANSWER 1 OF 1 CA COPYRIGHT 2005 ACS on STN  
AN **97:36514** CA  
TI Effect of adrenotropic substances on the growth and maturation of oocytes of the sea urchin, Strongylocentrotus nudus  
AU Khotimchenko, Yu. S.  
CS Far East Sci. Cent., Inst. Mar. Biol., Vladivostok, 690022, USSR  
SO Experientia (1982), 38(6), 696-7  
CODEN: EXPEAM; ISSN: 0014-4754  
DT Journal  
LA English

=> d ab

L3 ANSWER 1 OF 1 CA COPYRIGHT 2005 ACS on STN  
AB The oocyte size was decreased after the administration of the adrenomimetics, noradrenaline, dopamine, and ephedrine, to S. nudus. The administration of the adrenolytics, propranolol, oxyprenolol, and dihydroergotamine, caused an increase in the sea urchin oocyte size. Thus, oogenesis in the sea urchin may be regulated by a monoaminergic system.

=> log y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	15.51	15.72
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-2.04	-2.04

STN INTERNATIONAL LOGOFF AT 14:50:53 ON 12 SEP 2005

aerobic conditions. Within a 2 h time interval, leukocyte phagocytosis in the presence of these vasoactive compounds resulted in a reduction in bacterial count in the supernatant. Intracellular kill of the phagocytized **bacteria**, as exemplified by the reduction in the total count of the suspensions, was comparable in the suspensions containing vasoactive compounds and the controls.

ORGN Classifier

**Bacteria** 05000

Super Taxa

Microorganisms

Taxa Notes

**Bacteria**, Eubacteria, Microorganisms

ORGN Classifier

Leporidae 86040

Super Taxa

Lagomorpha; Mammalia; Vertebrata; Chordata; Animalia

Taxa Notes

Animals, Chordates, Lagomorphs, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Vertebrates

AB In rabbits, epinephrine altered the distribution of the i.v. injected fluorescein dye. At the epinephrine injection site the skin did not fluoresce, indicating that the dye had limited access to that tissue. The uninjected skin as well as the skin subjected to phentolamine fluoresced brightly. The minimal concentration of a phentolamine solution that inhibited epinephrine (1:100,000)-induced vasoconstriction was 1:50,000. Epinephrine potentiated the development of infection. When 6.0 + 10<sup>6</sup> Staphylococcus aureus were injected into skin in the presence of epinephrine, all sites developed infection as compared to only a 12.5% infection rate in the control wounds. The addition of phentolamine (1:50,000) to the epinephrine eliminated its damaging effects. The infection rate of contaminated tissue subjected to the mixture of vasoactive drugs did not differ significantly from that of controls. Phentolamine by itself did not **enhance** the incidence of infection. The infection rate of tissues was proportional to the magnitude of wound induration and the number of viable **bacteria** recovered from the wounds. Under aerobic conditions, the presence of epinephrine and phentolamine in the nutrient broth did not influence the **growth** of S. aureus in **vitro**. The bacterial counts of the suspensions containing the vasoactive compounds did not differ significantly from those of the control suspensions. Epinephrine and phentolamine did not interfere with in **vitro** leukocyte phagocytosis of **bacteria** and subsequent intracellular kill under aerobic conditions. Within a 2 h time interval, leukocyte phagocytosis in the presence of these vasoactive compounds resulted in a reduction in bacterial count in the supernatant. Intracellular kill of the phagocytized **bacteria**, as exemplified by the reduction in the total count of the suspensions, was comparable in the suspensions containing vasoactive compounds and the controls.

L13 ANSWER 6 OF 10 CAPLUS COPYRIGHT 2005 ACS on STN

TI Effect of copper on thyroxine potentiation of in **vitro** **epinephrine** action on smooth muscle

AN 1964:11057 CAPLUS

DN 60:11057

OREF 60:2016e-g

TI Effect of copper on thyroxine potentiation of in **vitro** **epinephrine** action on smooth muscle

AU Shida, H.; Meyers, M. A.; Barker, S. B.

CS Univ. of Alabama Med. Center, Birmingham

SO Journal of Pharmacology and Experimental Therapeutics (1963), 141(3), 280-4

CODEN: JPETAB; ISSN: 0022-3565

DT Journal

LA Unavailable

TI Effect of copper on thyroxine potentiation of in **vitro**  
**epinephrine** action on smooth muscle  
 SO Journal of Pharmacology and Experimental Therapeutics (1963),  
 141(3), 280-4  
 CODEN: JPETAB; ISSN: 0022-3565  
 AB Using the method of Furchgott and Bhadrakom (CA 47, 10111i) employing the  
 contraction of a helical aortic strip of a rabbit, it was confirmed that  
 in **vitro** addition of thyroxine (I) enhanced the contracting effect  
 of epinephrine and norepinephrine. The KrebsRinger NaHCO<sub>3</sub> glucose bath  
 solution contained varying contaminating levels of Cu. No **enhancing**  
 effect was observed at low Cu levels (1.5  $\gamma$ /l.) but was clearly  
 evident at higher levels (3.5 and 6.6  $\gamma$ /l.). Since  
 ethylenediaminetetraacetate behaved similarly to I, the effect was  
 probably due to chelation of the Cu in the working solution Zn and Fe salts  
 in higher concentration (100  $\gamma$ /l.) also suppressed catechol amine action  
 which could be reversed by I. Mn at 10  $\gamma$ /l. was as effective as Cu  
 but was not reversed by I.  
 IT **Viruses**  
 (poliomyelitis, ribonucleic acid of, base composition of)  
 AB Using the method of Furchgott and Bhadrakom (CA 47, 10111i) employing the  
 contraction of a helical aortic strip of a rabbit, it was confirmed that  
 in **vitro** addition of thyroxine (I) enhanced the contracting effect  
 of epinephrine and norepinephrine. The KrebsRinger NaHCO<sub>3</sub> glucose bath  
 solution contained varying contaminating levels of Cu. No **enhancing**  
 effect was observed at low Cu levels (1.5  $\gamma$ /l.) but was clearly  
 evident at higher levels (3.5 and 6.6  $\gamma$ /l.). Since  
 ethylenediaminetetraacetate behaved similarly to I, the effect was  
 probably due to chelation of the Cu in the working solution Zn and Fe salts  
 in higher concentration (100  $\gamma$ /l.) also suppressed catechol amine action  
 which could be reversed by I. Mn at 10  $\gamma$ /l. was as effective as Cu  
 but was not reversed by I.  
 L13 ANSWER 7 OF 10 MEDLINE on STN  
 TI Herpes simplex **virus** recovery in neural tissues after ocular HSV  
 shedding induced by **epinephrine** iontophoresis to the rabbit  
 cornea.  
 AN 83134751 MEDLINE  
 DN PubMed ID: 6298139  
 TI Herpes simplex **virus** recovery in neural tissues after ocular HSV  
 shedding induced by **epinephrine** iontophoresis to the rabbit  
 cornea.  
 AU Hill J M; Kwon B S; Shimomura Y; Colborn G L; Yaghmai F; Gangarosa L P  
 NC NEI-EY-03331 (NEI)  
 NIDR-DE-04917 (NIDCR)  
 SO Investigative ophthalmology & visual science, (1983 Feb) 24 (2)  
 243-7.  
 Journal code: 7703701. ISSN: 0146-0404.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 198304  
 ED Entered STN: 19900318  
 Last Updated on STN: 20000303  
 Entered Medline: 19830415  
 TI Herpes simplex **virus** recovery in neural tissues after ocular HSV  
 shedding induced by **epinephrine** iontophoresis to the rabbit  
 cornea.  
 SO Investigative ophthalmology & visual science, (1983 Feb) 24 (2)  
 243-7.  
 Journal code: 7703701. ISSN: 0146-0404.  
 AB Ocular HSV-1 shedding from latently infected rabbits was induced by  
 iontophoresis of 0.01% epinephrine into the eye. Anodal Iontophoresis of  
 epinephrine was performed at 0.8 mAmps for 8 min once a day for 3

consecutive days. Shedding was determined by the presence of HSV-1 in the preocular tear film obtained via eye swabs. Bilateral epinephrine iontophoresis performed on selected days during 220-280 days after inoculation resulted in HSV-1 shedding in 75% of the eyes (30/40) and 100% of the rabbits (20/20). Following the induction of ocular HSV-1 shedding, rabbits were killed and selected neural tissues were homogenized. Cell-free preparations were assayed for the presence of infectious virions using primary rabbit kidney cell monolayers. When the tissues were homogenized immediately after death, **virus** was detected in only one neural tissue, the trigeminal ganglia. However, when the tissues were incubated *in vitro* for 18-24 hours prior to the homogenization, infectious HSV-1 was recovered from homogenates of the trigeminal ganglion, superior cervical ganglion, the ophthalmic branch of the trigeminal nerve, and the root entry zone of the trigeminal nerve. A relationship was noted between the time of the last ocular shedding and recovery of infectious HSV from the tissue homogenates. Furthermore, a positive correlation in 11 eyes between the recovery of HSV-1 from the perocular tear film and HSV-1 recovery from one or more corresponding neural tissues was found. These results suggested that epinephrine iontophoresis to the cornea triggered an "alteration" in the state of the **virus** in the neural tissues of the latently infected rabbits and that the change can be related to the induced ocular shedding.

CT

Animals

Cornea: MI, microbiology

**Culture Techniques**

\*Epinephrine: AD, administration & dosage

\*Iontophoresis

\*Keratitis, Dendritic: MI, microbiology

\*Nerve Tissue: MI, microbiology

Rabbits

Research Support, U.S. Gov't, P.H.S.

**\*Simplexvirus: GD, growth & development**

Tears: MI, microbiology

Time Factors

Virion: IP, isolation & purification

**\*Virus Activation**

AB

Ocular HSV-1 shedding from latently infected rabbits was induced by iontophoresis of 0.01% epinephrine into the eye. Anodal Iontophoresis of epinephrine was performed at 0.8 mAmps for 8 min once a day for 3 consecutive days. Shedding was determined by the presence of HSV-1 in the preocular tear film obtained via eye swabs. Bilateral epinephrine iontophoresis performed on selected days during 220-280 days after inoculation resulted in HSV-1 shedding in 75% of the eyes (30/40) and 100% of the rabbits (20/20). Following the induction of ocular HSV-1 shedding, rabbits were killed and selected neural tissues were homogenized. Cell-free preparations were assayed for the presence of infectious virions using primary rabbit kidney cell monolayers. When the tissues were homogenized immediately after death, **virus** was detected in only one neural tissue, the trigeminal ganglia. However, when the tissues were incubated *in vitro* for 18-24 hours prior to the homogenization, infectious HSV-1 was recovered from homogenates of the trigeminal ganglion, superior cervical ganglion, the ophthalmic branch of the trigeminal nerve, and the root entry zone of the trigeminal nerve. A relationship was noted between the time of the last ocular shedding and recovery of infectious HSV from the tissue homogenates. Furthermore, a positive correlation in 11 eyes between the recovery of HSV-1 from the perocular tear film and HSV-1 recovery from one or more corresponding neural tissues was found. These results suggested that epinephrine iontophoresis to the cornea triggered an "alteration" in the state of the **virus** in the neural tissues of the latently infected rabbits and that the change can be related to the induced ocular shedding.

L13 ANSWER 8 OF 10 USPATFULL on STN

TI Method employing gonadal hormones and **dopamine** agonist  
intended for combined use in the improvement of lymphocyte function  
AN 90:98709 USPATFULL  
TI Method employing gonadal hormones and **dopamine** agonist  
intended for combined use in the improvement of lymphocyte function  
IN Smith, R. Arnold, Jackson, MS, United States  
PA McAdory, George D., Jackson, MS, United States (U.S. individual)  
PI US 4980358 19901225 <--  
AI US 1990-494327 19900316 (7)  
RLI Continuation-in-part of Ser. No. US 1988-177121, filed on 4 Apr 1988,  
now patented, Pat. No. US 4929640  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Friedman, Stanley J.; Assistant Examiner: Criares, T.  
J.  
LREP Epstein, Edell & Retzer  
CLMN Number of Claims: 31  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 498  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L13 ANSWER 9 OF 10 USPATFULL on STN

TI **Dopamine**- $\beta$ -hydroxylase inhibitors  
AN 88:48763 USPATFULL  
TI **Dopamine**- $\beta$ -hydroxylase inhibitors  
IN Finkelstein, Joseph A., Philadelphia, PA, United States  
Kruse, Lawrence I., Haddonfield, NJ, United States  
Leonard, Thomas B., Haverford, PA, United States  
PA SmithKline Beckman Corporation, Philadelphia, PA, United States (U.S.  
corporation)  
PI US 4761415 19880802 <--  
AI US 1986-901120 19860828 (6)  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Gerstl, Robert; Assistant Examiner: Shen, Cecilia  
LREP Fabiano, Vincent L., Suter, Stuart R., Lourie, Alan D.  
CLMN Number of Claims: 15  
ECL Exemplary Claim: 1,11,15  
DRWN No Drawings  
LN.CNT 562  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L13 ANSWER 10 OF 10 USPATFULL on STN

TI **Dopamine**- $\beta$ -hydroxylase inhibitors  
AN 86:69756 USPATFULL  
TI **Dopamine**- $\beta$ -hydroxylase inhibitors  
IN Finkelstein, Joseph A., Philadelphia, PA, United States  
Kaiser, Carl, Haddon Heights, NJ, United States  
Kruse, Lawrence I., Haddonfield, NJ, United States  
PA SmithKline Beckman Corporation, Philadelphia, PA, United States (U.S.  
corporation)  
PI US 4628059 19861209 <--  
AI US 1985-793513 19851031 (6)  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Bond, Robert T.  
LREP Fabiano, Vincent L., Suter, Stuart R., Lourie, Alan D.  
CLMN Number of Claims: 21  
ECL Exemplary Claim: 1,15  
DRWN No Drawings  
LN.CNT 604



CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> log hold

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

69.16

203.62

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE

TOTAL

ENTRY

SESSION

CA SUBSCRIBER PRICE

-0.73

-2.77

SESSION WILL BE HELD FOR 60 MINUTES

STN INTERNATIONAL SESSION SUSPENDED AT 13:55:48 ON 12 SEP 2005

were preincubated with melanin, thus suggesting that interaction of melanin with viral proteins is an important aspect of the antiviral mechanism. These results make synthetic soluble melanins interesting candidates for further study as possible anti-HIV-1 therapeutics.

L13 ANSWER 3 OF 10 TOXCENTER COPYRIGHT 2005 ACS on STN DUPLICATE 3  
TI MANGANESE II UNCOUPLING OF THE **CATECHOLAMINE** SENSITIVE ADENYLATE  
CYCLASE SYSTEM OF RAT RETICULOCYTES PARALLEL EFFECTS ON CHOLERA TOXIN  
CATALYZED ADP RIBOSYLATION OF THE SYSTEM  
AN 1982:68444 TOXCENTER  
CP Copyright (c) 2005 The Thomson Corporation  
DN PREV198273070979  
TI MANGANESE II UNCOUPLING OF THE **CATECHOLAMINE** SENSITIVE ADENYLATE  
CYCLASE SYSTEM OF RAT RETICULOCYTES PARALLEL EFFECTS ON CHOLERA TOXIN  
CATALYZED ADP RIBOSYLATION OF THE SYSTEM  
AU LIMBIRD L E [Reprint author]; MACMILLAN S T  
CS DEP PHARMACOL, SCH MED, VANDERBILT UNIV, NASHVILLE, TENN 37232, USA  
SO Biochimica et Biophysica Acta, (1981) Vol. 677, No. 3-4, pp.  
408-416.  
CODEN: BBACAQ. ISSN: 0006-3002.  
DT Article  
FS BIOSIS  
OS BIOSIS 1982:210995  
LA ENGLISH  
ED Entered STN: 20011116  
Last Updated on STN: 20011116  
TI MANGANESE II UNCOUPLING OF THE **CATECHOLAMINE** SENSITIVE ADENYLATE  
CYCLASE SYSTEM OF RAT RETICULOCYTES PARALLEL EFFECTS ON CHOLERA TOXIN  
CATALYZED ADP RIBOSYLATION OF THE SYSTEM  
SO Biochimica et Biophysica Acta, (1981) Vol. 677, No. 3-4, pp.  
408-416.  
CODEN: BBACAQ. ISSN: 0006-3002.  
AB High concentrations of Mn<sup>2+</sup> interfere with functional interactions between  
the GTP-binding regulatory protein (G) and the catalytic moiety (C) of  
adenylate cyclase without perturbing interactions between receptor (R) and  
component G in rat reticulocyte membranes. The ability of cholera toxin  
to ADP-ribosylate component G and to **enhance** GTP-stimulated  
adenylate cyclase activity also appears to be correlated with the efficacy  
of the communication of component G with the adenylylase system.  
Thus, increasing the concentration of Mn<sup>2+</sup> in rat reticulocyte membrane  
during *in vitro* incubations causes a parallel loss of  
Gpp(NH)p-stimulated adenylylase activity, cholera toxin-catalyzed  
[32P]ADP-ribosylation of the 42,000 MW subunit of component G and cholera  
toxin-catalyzed enhancement of GTP-sensitive adenylylase activity.  
Removal of Mn<sup>2+</sup> by washing the membranes completely restores the  
sensitivity of adenylylase to all effectors, including cholera  
toxin. Exposure of membranes to Mn<sup>2+</sup> apparently provides a useful tool  
for reversibly uncoupling catecholamine-sensitive adenylylase  
systems. The extent of cholera toxin-catalyzed ADP-ribosylation of  
membrane substrates, i.e., the G component may rely on functional  
communication among the various components of the adenylylase  
system. A corollary of the latter is that the amount of  
[32P]ADP-ribose-product detected in a membrane may reflect both the  
quantity and coupling efficiency of component G.  
ORGN Classifier  
Vibrionaceae 06704  
Super Taxa  
Facultatively Anaerobic Gram-Negative Rods; Eubacteria; Bacteria;  
Microorganisms  
Taxa Notes  
**Bacteria**, Eubacteria, Microorganisms  
ORGN Classifier  
Muridae 86375  
Super Taxa

Rodentia; Mammalia; Vertebrata; Chordata; Animalia

Taxa Notes

Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates

AB High concentrations of Mn<sup>2+</sup> interfere with functional interactions between the GTP-binding regulatory protein (G) and the catalytic moiety (C) of adenylate cyclase without perturbing interactions between receptor (R) and component G in rat reticulocyte membranes. The ability of cholera toxin to ADP-ribosylate component G and to **enhance** GTP-stimulated adenylate cyclase activity also appears to be correlated with the efficacy of the communication of component G with the adenylate cyclase system. Thus, increasing the concentration of Mn<sup>2+</sup> in rat reticulocyte membrane during *in vitro* incubations causes a parallel loss of Gpp(NH)p-stimulated adenylate cyclase activity, cholera toxin-catalyzed [<sup>32</sup>P]ADP-ribosylation of the 42,000 MW subunit of component G and cholera toxin-catalyzed enhancement of GTP-sensitive adenylate cyclase activity. Removal of Mn<sup>2+</sup> by washing the membranes completely restores the sensitivity of adenylate cyclase to all effectors, including cholera toxin. Exposure of membranes to Mn<sup>2+</sup> apparently provides a useful tool for reversibly uncoupling catecholamine-sensitive adenylate cyclase systems. The extent of cholera toxin-catalyzed ADP-ribosylation of membrane substrates, i.e., the G component may rely on functional communication among the various components of the adenylate cyclase system. A corollary of the latter is that the amount of [<sup>32</sup>P]ADP-ribose-product detected in a membrane may reflect both the quantity and coupling efficiency of component G.

L13 ANSWER 4 OF 10 TOXCENTER COPYRIGHT 2005 ACS on STN DUPLICATE 4

TI EFFECTS OF **DOPAMINE** ON PROLACTIN SECRETION AND CYCLIC AMP  
ACCUMULATION IN THE RAT ANTERIOR PITUITARY GLAND

AN 1981:69281 TOXCENTER

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DN PREV198171082911

TI EFFECTS OF **DOPAMINE** ON PROLACTIN SECRETION AND CYCLIC AMP  
ACCUMULATION IN THE RAT ANTERIOR PITUITARY GLAND

AU RAY K P [Reprint author]; WALLIS M

CS SCH BIOL SCI, UNIV SUSSEX, FALMER, BRIGHTON BN1 9QG, ENGL, UK

SO Biochemical Journal, (1981) Vol. 194, No. 1, pp. 119-128.  
ISSN: 0264-6021.

DT Article

FS BIOSIS

OS BIOSIS 1981:212919

LA ENGLISH

ED Entered STN: 20011116

Last Updated on STN: 20011116

TI EFFECTS OF **DOPAMINE** ON PROLACTIN SECRETION AND CYCLIC AMP  
ACCUMULATION IN THE RAT ANTERIOR PITUITARY GLAND

SO Biochemical Journal, (1981) Vol. 194, No. 1, pp. 119-128.  
ISSN: 0264-6021.

AB The effects of dopamine on pituitary prolactin secretion and pituitary cAMP accumulation were studied by using anterior pituitary glands from adult female rats, incubated *in vitro*. During 2 h incubations, significant inhibition of prolactin secretion was achieved at concentrations between 1 and 10 nM-dopamine. However, 0.1-1  $\mu$ M-dopamine was required before a significant decrease in pituitary cAMP content was observed. In the presence of 1  $\mu$ M-dopamine, pituitary cAMP content decreased rapidly to reach about 75% of the control value within 20 min, and there was no further decrease for at least 2 h. Incubation with the phosphodiesterase inhibitors, theophylline (8 mM) or isobutylmethylxanthine (2 mM), increased pituitary cAMP concentrations 3- and 6-fold, respectively. Dopamine (1  $\mu$ M) had no effect on the cAMP accumulation measured in the presence of theophylline, but inhibited the isobutylmethylxanthine-induced increase by 50%. The dopamine inhibition of prolactin secretion was not affected by either inhibitor. Two

TI Novel nerve **growth** factor-responsiveness of **catecholamine** biosynthesis and secretion in clonal rat pheochromocytoma cells cultured in a hormone-supplemented serum-free medium

L9 ANSWER 34 OF 44 CA COPYRIGHT 2005 ACS on STN  
 TI Plasticity of pheochromocytoma (PC12) cells demonstrated by nerve **growth** factor or glucocorticoid treatment: a **catecholamine** fluorescence and electron microscopic investigation

L9 ANSWER 35 OF 44 CA COPYRIGHT 2005 ACS on STN  
 TI Immunohistochemical and immunocytochemical localization of myosin, chromogranin A and **dopamine**- $\beta$ -hydroxylase in nerve cells in **culture** and in adrenal glands

L9 ANSWER 36 OF 44 CA COPYRIGHT 2005 ACS on STN  
 TI Biochemical characterization of **norepinephrine**-3H uptake in dissociated brain **cell** cultures from chick embryos

L9 ANSWER 37 OF 44 CA COPYRIGHT 2005 ACS on STN  
 TI A clonal rat pheochromocytoma **cell** line possesses synthesizing ability of  $\gamma$ -aminobutyric acid together with **catecholamine** and acetylcholine

L9 ANSWER 38 OF 44 CA COPYRIGHT 2005 ACS on STN  
 TI **Catecholamine**-containing neurons from rat brain in **culture**: response to peripheral and central target tissues

L9 ANSWER 39 OF 44 CA COPYRIGHT 2005 ACS on STN  
 TI Role of nerve **growth** factor in the development of rat sympathetic neurons in vitro. I. Survival, **growth**, and differentiation of **catecholamine** production

L9 ANSWER 40 OF 44 CA COPYRIGHT 2005 ACS on STN  
 TI Selective induction of tyrosine hydroxylase and **dopamine**  $\beta$ -hydroxylase by nerve **growth** factor: comparison between adrenal medulla and sympathetic ganglia of adult and newborn rats

L9 ANSWER 41 OF 44 CA COPYRIGHT 2005 ACS on STN  
 TI Changes in **catecholamine** synthesizing enzyme activities during neuronal **growth** and degeneration

L9 ANSWER 42 OF 44 CA COPYRIGHT 2005 ACS on STN  
 TI Production of **catecholamine**-containing cells in vitro by young chick embryos studied by the histochemical fluorescence method

L9 ANSWER 43 OF 44 CA COPYRIGHT 2005 ACS on STN  
 TI Production of **catecholamine**-containing cells in vitro by young chick embryos. Effects of nerve **growth** factor (NGF) and its antiserum

L9 ANSWER 44 OF 44 CA COPYRIGHT 2005 ACS on STN  
 TI Influence of pituitary hormones and **norepinephrine** on the size of adipose cells in organ **culture**

=> d ti bib hit ab 23,24, 28

L9 ANSWER 23 OF 44 CA COPYRIGHT 2005 ACS on STN  
 TI **Catecholamine** modulation of embryonic palate mesenchymal **cell** DNA synthesis  
 AN 104:82581 CA  
 TI **Catecholamine** modulation of embryonic palate mesenchymal **cell** DNA synthesis

AU Pisano, M. Michele; Schneiderman, Martin H.; Greene, Robert M.  
 CS Daniel Baugh Inst., Thomas Jefferson Univ., Philadelphia, PA, 19107, USA  
 SO Journal of Cellular Physiology (1986), 126(1), 84-92  
 CODEN: JCLLAX; ISSN: 0021-9541

DT Journal  
 LA English

TI **Catecholamine** modulation of embryonic palate mesenchymal  
**cell** DNA synthesis

SO Journal of Cellular Physiology (1986), 126(1), 84-92  
 CODEN: JCLLAX; ISSN: 0021-9541

AB By utilizing quiescent populations of murine embryonic palate mesenchymal (MEPM) cells in vitro, hormonal regulation of palatal **cell** proliferation was examined MEPM cells in **culture** were rendered quiescent by 48 h serum deprivation and were subsequently released from **growth** arrest by readdn. of medium containing 10% (volume/volume) serum. The progression of cells into S-phase of the **cell** cycle was monitored by autoradiog. anal. of tritiated thymidine incorporation. Palate mesenchymal **cell** entry into S-phase was preceded by a 6-8-h prereplicative lag period, after which time DNA synthesis increased and cells reached a maximum labeling index by 22 h. Addition of 10  $\mu$ M ( $\pm$ )-isoproterenol [149-53-1] to **cell** cultures at the time of release from **growth** arrest lengthened the prereplicative lag period and delayed cellular entry into S-phase by an addnl. 2-4 h. The rate of cellular progression through S-phase remained unaltered. The inhibitory effect of isoproterenol on the initiation of MEPM **cell** DNA synthesis was abolished by pretreatment of cells with propranolol at a concentration (100  $\mu$ M) that prevented isoproterenol-induced elevations of cAMP [60-92-4]. Addition of PGE2 [363-24-6] to **cell** cultures, at a concentration that markedly stimulates cAMP formation, mimicked the inhibitory effect of isoproterenol on cellular progression into S-phase. Evidently, the  $\beta$ -adrenergic catecholamine isoproterenol modulates MEPM **cell** proliferation in vitro via a receptor-mediated mechanism, and the delayed initiation of DNA synthesis in these cells is a cAMP-dependent phenomenon.

IT **Cell cycle**  
 (S-phase, DNA formation by palate of embryo response to catecholamine in relation to)

AB By utilizing quiescent populations of murine embryonic palate mesenchymal (MEPM) cells in vitro, hormonal regulation of palatal **cell** proliferation was examined MEPM cells in **culture** were rendered quiescent by 48 h serum deprivation and were subsequently released from **growth** arrest by readdn. of medium containing 10% (volume/volume) serum. The progression of cells into S-phase of the **cell** cycle was monitored by autoradiog. anal. of tritiated thymidine incorporation. Palate mesenchymal **cell** entry into S-phase was preceded by a 6-8-h prereplicative lag period, after which time DNA synthesis increased and cells reached a maximum labeling index by 22 h. Addition of 10  $\mu$ M ( $\pm$ )-isoproterenol [149-53-1] to **cell** cultures at the time of release from **growth** arrest lengthened the prereplicative lag period and delayed cellular entry into S-phase by an addnl. 2-4 h. The rate of cellular progression through S-phase remained unaltered. The inhibitory effect of isoproterenol on the initiation of MEPM **cell** DNA synthesis was abolished by pretreatment of cells with propranolol at a concentration (100  $\mu$ M) that prevented isoproterenol-induced elevations of cAMP [60-92-4]. Addition of PGE2 [363-24-6] to **cell** cultures, at a concentration that markedly stimulates cAMP formation, mimicked the inhibitory effect of isoproterenol on cellular progression into S-phase. Evidently, the  $\beta$ -adrenergic catecholamine isoproterenol modulates MEPM **cell** proliferation in vitro via a receptor-mediated mechanism, and the delayed initiation of DNA synthesis in these cells is a cAMP-dependent phenomenon.

L9 ANSWER 24 OF 44 CA COPYRIGHT 2005 ACS on STN  
 TI Relationship between **dopamine** content and its secretion in PC12

cells as a function of **cell growth**

AN 103:207450 CA

TI Relationship between **dopamine** content and its secretion in PC12 cells as a function of **cell growth**

AU Takashima, Akihiko; Koike, Tatsuro

CS Dep. Nat. Sci., Saga Med. Sch., Nabeshima, 840-01, Japan

SO Biochimica et Biophysica Acta (1985), 847(1), 101-7  
CODEN: BBACAQ; ISSN: 0006-3002

DT Journal

LA English

TI Relationship between **dopamine** content and its secretion in PC12 cells as a function of **cell growth**

SO Biochimica et Biophysica Acta (1985), 847(1), 101-7  
CODEN: BBACAQ; ISSN: 0006-3002

AB The relation between dopamine [51-61-6] biosynthesis and its stimulus-induced secretion was studied in PC12 cells as a function of **cell growth**. The endogenous dopamine content depended on **cell growth**, and reached a maximum in the stationary phase. This increase was associated both with an increase in the specific activity of tyrosine 3-monooxygenase [9036-22-0], and with an increase of DOPA-decarboxylase [9042-64-2] in the cells. On the other hand, the maximal release of dopamine occurred in the late exponential phase before the endogenous dopamine was maximally synthesized in the cells. Moreover, the uptake of  $45\text{Ca}^{2+}$  stimulated with either carbamylcholine [462-58-8] or high  $\text{K}^+$  was also regulated by **cell division**: the maximal uptake took place in the same period of **culture** in which the maximal release of dopamine was observed. Thus, the biosynthesis and secretion of dopamine are sep. regulated in PC12 cells.

ST pheochromocytoma dopamine metab **cell division**

IT Biological transport  
(of calcium, by pheochromocytoma, **cell division** and dopamine formation and release in relation to)

IT Pheochromocytoma  
(PC12, dopamine formation and secretion by, **cell division** in relation to)

IT **Cell division**  
(mitosis, by pheochromocytoma, dopamine formation and release in relation to)

IT 462-58-8 7440-09-7, biological studies  
RL: BIOL (Biological study)  
(calcium uptake by pheochromocytoma stimulation by, **cell division** and dopamine release in relation to)

IT 51-61-6, biological studies  
RL: BIOL (Biological study)  
(formation and secretion of, by pheochromocytoma, **cell division** in relation to)

IT 7440-70-2, biological studies  
RL: BIOL (Biological study)  
(uptake of, by pheochromocytoma, **cell division** and dopamine release in relation to)

AB The relation between dopamine [51-61-6] biosynthesis and its stimulus-induced secretion was studied in PC12 cells as a function of **cell growth**. The endogenous dopamine content depended on **cell growth**, and reached a maximum in the stationary phase. This increase was associated both with an increase in the specific activity of tyrosine 3-monooxygenase [9036-22-0], and with an increase of DOPA-decarboxylase [9042-64-2] in the cells. On the other hand, the maximal release of dopamine occurred in the late exponential phase before the endogenous dopamine was maximally synthesized in the cells. Moreover, the uptake of  $45\text{Ca}^{2+}$  stimulated with either carbamylcholine [462-58-8] or high  $\text{K}^+$  was also regulated by **cell division**: the maximal uptake took place in the same period of **culture** in which the maximal release of dopamine was observed. Thus, the biosynthesis and secretion of dopamine are sep. regulated in PC12 cells.

of prolactin secretion was not affected by either inhibitor. Two derivatives of cAMP (dibutyryl cAMP and 8-bromo cAMP) were unable to block the dopamine (1  $\mu$ M) inhibition of prolactin secretion, although 8-bromo cAMP (2mM) significantly stimulated prolactin secretion and both compounds increased **growth** hormone [GH] release. Cholera toxin (3  $\mu$ g/ml for 4 h) increased pituitary cAMP concentrations 4-to 5-fold, but had no effect on prolactin secretion. The inhibition of prolactin secretion by dopamine was unaffected by cholera toxin, despite the fact that dopamine had no effect on the raised pituitary cAMP concentration caused by this factor. Dopamine had no significant effect on either basal or stimulated GH secretion under any of the conditions tested. The inhibitory effects of dopamine on prolactin secretion are probably not mediated by lowering of cAMP concentration, although modulation of the concentration of this nucleotide, in some other circumstances, may alter the secretion of the hormone.

L13 ANSWER 5 OF 10 TOXCENTER COPYRIGHT 2005 ACS on STN DUPLICATE 5  
 TI STUDIES OF THE MECHANISMS BY WHICH **EPINEPHRINE** DAMAGES TISSUE DEFENSES  
 AN 1978:55907 TOXCENTER  
 CP Copyright (c) 2005 The Thomson Corporation  
 DN PREV197865074388  
 TI STUDIES OF THE MECHANISMS BY WHICH **EPINEPHRINE** DAMAGES TISSUE DEFENSES  
 AU MAGEE C [Reprint author]; RODEHEAVER G T; EDGERTON M T; GOLDEN G T; HAURY B; EDLICH R F  
 CS DEP PLAST SURG, UNIV VA SCH MED, CHARLOTTESVILLE, VA 22901, USA  
 SO Journal of Surgical Research, (1977) Vol. 23, No. 2, pp. 126-131.  
 CODEN: JSGRA2. ISSN: 0022-4804.  
 DT Article  
 FS BIOSIS  
 OS BIOSIS 1978:187388  
 LA ENGLISH  
 ED Entered STN: 20011116  
 Last Updated on STN: 20011116  
 TI STUDIES OF THE MECHANISMS BY WHICH **EPINEPHRINE** DAMAGES TISSUE DEFENSES  
 SO Journal of Surgical Research, (1977) Vol. 23, No. 2, pp. 126-131.  
 CODEN: JSGRA2. ISSN: 0022-4804.  
 AB In rabbits, epinephrine altered the distribution of the i.v. injected fluorescein dye. At the epinephrine injection site the skin did not fluoresce, indicating that the dye had limited access to that tissue. The uninjected skin as well as the skin subjected to phentolamine fluoresced brightly. The minimal concentration of a phentolamine solution that inhibited epinephrine (1:100,000)-induced vasoconstriction was 1:50,000. Epinephrine potentiated the development of infection. When  $6.0 \times 10^6$  Staphylococcus aureus were injected into skin in the presence of epinephrine, all sites developed infection as compared to only a 12.5% infection rate in the control wounds. The addition of phentolamine (1:50,000) to the epinephrine eliminated its damaging effects. The infection rate of contaminated tissue subjected to the mixture of vasoactive drugs did not differ significantly from that of controls. Phentolamine by itself did not **enhance** the incidence of infection. The infection rate of tissues was proportional to the magnitude of wound induration and the number of viable **bacteria** recovered from the wounds. Under aerobic conditions, the presence of epinephrine and phentolamine in the nutrient broth did not influence the **growth** of S. aureus in **vitro**. The bacterial counts of the suspensions containing the vasoactive compounds did not differ significantly from those of the control suspensions. Epinephrine and phentolamine did not interfere with in **vitro** leukocyte phagocytosis of **bacteria** and subsequent intracellular kill under

derivatives of cAMP (dibutyryl cAMP and 8-bromo cAMP) were unable to block the dopamine (1  $\mu$ M) inhibition of prolactin secretion, although 8-bromo cAMP (2mM) significantly stimulated prolactin secretion and both compounds increased growth hormone [GH] release. Cholera toxin (3  $\mu$ g/ml for 4 h) increased pituitary cAMP concentrations 4-to 5-fold, but had no effect on prolactin secretion. The inhibition of prolactin secretion by dopamine was unaffected by cholera toxin, despite the fact that dopamine had no effect on the raised pituitary cAMP concentration caused by this factor. Dopamine had no significant effect on either basal or stimulated GH secretion under any of the conditions tested. The inhibitory effects of dopamine on prolactin secretion are probably not mediated by lowering of cAMP concentration, although modulation of the concentration of this nucleotide, in some other circumstances, may alter the secretion of the hormone.

ST Major Concepts

Endocrine System (Chemical Coordination and Homeostasis); Metabolism; Nervous System (Neural Coordination); Pharmacology

ST Miscellaneous Descriptors

AUTONOMIC-DRUG THEOPHYLLINE ISO BUTYLMETHYL XANTHINE ENZYME  
INHIBITOR-DRUG DI BUTYRYL CYCLIC AMP 8 BROMO CYCLIC AMP CHOLERA TOXIN  
METABOLIC-DRUG PHOSPHO DI ESTERASE GROWTH HORMONE  
PHARMACODYNAMICS

ORGN Classifier

Vibrionaceae 06704

Super Taxa

Facultatively Anaerobic Gram-Negative Rods; Eubacteria; Bacteria; Microorganisms

Taxa Notes

**Bacteria**, Eubacteria, Microorganisms

ORGN Classifier

Theaceae 26845

Super Taxa

Dicotyledones; Angiospermae; Spermatophyta; Plantae

Taxa Notes

Angiosperms, Dicots, Plants, Spermatophytes, Vascular Plants

ORGN Classifier

Muridae 86375

Super Taxa

Rodentia; Mammalia; Vertebrata; Chordata; Animalia

Taxa Notes

Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates

RN

51-61-6 (DOPAMINE)  
9002-62-4 (PROLACTIN)  
60-92-4 (CYCLIC AMP)  
58-55-9 (THEOPHYLLINE)  
28822-58-4 (ISOBUTYLMETHYLXANTHINE)  
362-74-3 (DIBUTYRYL CYCLIC AMP)  
9025-82-5 (PHOSPHODIESTERASE)  
9002-72-6 (GROWTH HORMONE)

AB

The effects of dopamine on pituitary prolactin secretion and pituitary cAMP accumulation were studied by using anterior pituitary glands from adult female rats, incubated *in vitro*. During 2 h incubations, significant inhibition of prolactin secretion was achieved at concentrations between 1 and 10 nM-dopamine. However, 0.1-1 $\mu$ M-dopamine was required before a significant decrease in pituitary cAMP content was observed. In the presence of 1  $\mu$ M-dopamine, pituitary cAMP content decreased rapidly to reach about 75% of the control value within 20 min, and there was no further decrease for at least 2 h. Incubation with the phosphodiesterase inhibitors, theophylline (8 mM) or isobutylmethylxanthine (2mM), increased pituitary cAMP concentrations 3- and 6-fold, respectively. Dopamine (1  $\mu$ M) had no effect on the cAMP accumulation measured in the presence of theophylline, but inhibited the isobutylmethylxanthine-induced increase by 50%. The dopamine inhibition



**dopamine**-induced neurotoxicity in human and rodent neuronal cultures: possible implications for Parkinson's disease

```
=> s l6 and @py<1991
'1991' NOT A VALID FIELD CODE
      0 @PY<1991
L7      0 L6 AND @PY<1991
```

```
=> s l6 and @py=<1991
'1991' NOT A VALID FIELD CODE
      0 @PY=<1991
L8      0 L6 AND @PY=<1991
```

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=> s l6 and py=<1991
      14083918 PY=<1991
L9      44 L6 AND PY=<1991
```

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=> d his
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(FILE 'HOME' ENTERED AT 13:22:16 ON 12 SEP 2005)

FILE 'CAPLUS' ENTERED AT 13:22:39 ON 12 SEP 2005

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L1      0 S DYER/AU AND VOL AND 102 OR MOGER/AU AND VOL 97 AND 1982
L2      0 S DYER AND VOL AND 102 OR MOGER AND VOL 97 AND 1982
L3      0 S DYER.INV AND VOL AND 102 OR MOGER.INV AND VOL 97 AND 1982
      E DYER
```

FILE 'CA' ENTERED AT 13:26:08 ON 12 SEP 2005

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L4      0 S (DYER/INV OR DYER/AU) AND VOL 102 AND 1985
      E DYER
L5      490 S (GROWTH OR ENHANCE OR ENHANCING) AND (CATECHOLAMINE OR EPINEP
L6      105 S (GROWTH OR ENHANCE OR ENHANCING) AND (CATECHOLAMINE OR EPINEP
L7      0 S L6 AND @PY<1991
L8      0 S L6 AND @PY=<1991
L9      44 S L6 AND PY=<1991
```

```
=> d ti 1-44
```

```
L9 ANSWER 1 OF 44 CA COPYRIGHT 2005 ACS on STN
TI Angiotensin II increases catecholamine release from bovine
adrenal medulla but does not enhance that evoked by potassium
depolarization or by carbachol
```

```
L9 ANSWER 2 OF 44 CA COPYRIGHT 2005 ACS on STN
TI Effect of dopamine and bromocriptine on secretion of
growth hormone by pituitary growth hormone secreting
tumor in cell culture
```

```
L9 ANSWER 3 OF 44 CA COPYRIGHT 2005 ACS on STN
TI Serotonin and nialamide differentially regulate survival and
growth of cultured serotonin and catecholamine neurons
```

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L9 ANSWER 4 OF 44 CA COPYRIGHT 2005 ACS on STN
TI Pertussis toxin stimulation of catecholamine release from
adrenal medullary chromaffin cells: mechanism may be by direct activation
of L-type and G-type calcium channels
```

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L9 ANSWER 5 OF 44 CA COPYRIGHT 2005 ACS on STN
TI Growth of cultured human cerebral meningiomas is inhibited by
dopaminergic agents. Presence of high affinity dopamine-D1
receptors
```

```
L9 ANSWER 6 OF 44 CA COPYRIGHT 2005 ACS on STN
```

L6 ANSWER 1 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI Method of in vitro differentiation of neural stem cells, motor neurons, and **dopamine** neurons from primate embryonic stem cells

L6 ANSWER 2 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI Transactivation of epidermal **growth** factor receptor mediates **catecholamine**-induced **growth** of vascular smooth muscle

L6 ANSWER 3 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI Does acetylcholinesterase inhibition affect **catecholamine** secretion by adrenomedullary cells?

L6 ANSWER 4 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI Role of calcium in neurotensin-evoked enhancement in firing in mesencephalic **dopamine** neurons

L6 ANSWER 5 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI **Dopamine** Agonist 3-PPP Fails to Protect Against MPTP-Induced Toxicity

L6 ANSWER 6 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI Myocyte contractile activity modulates **norepinephrine** cytotoxicity and survival effects of neuregulin-1 $\beta$

L6 ANSWER 7 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI **Catecholamine**-induced vascular wall **growth** is dependent on generation of reactive oxygen species

L6 ANSWER 8 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI Effects of **dopamine** agonists bromocriptine, pergolide, cabergoline, and SKF-38393 on GDNF, NGF, and BDNF synthesis in cultured mouse astrocytes

L6 ANSWER 9 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI Role of **epinephrine** stimulation of CNS  $\alpha$ 1-adrenoceptors in motor activity in mice

L6 ANSWER 10 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI Interactions of cyclic adenosine monophosphate, brain-derived neurotrophic factor, and glial **cell** line-derived neurotrophic factor treatment on the survival and **growth** of postnatal mesencephalic **dopamine** neurons in vitro

L6 ANSWER 11 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI D1 **dopamine** receptor regulation of **cell** cycle in FGF- and EGF-supported primary cultures of embryonic cerebral cortical precursor cells

L6 ANSWER 12 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI A site-specific mutation of tyrosine hydroxylase reduces feedback inhibition by **dopamine** in genetically modified cells grafted in parkinsonian rats

L6 ANSWER 13 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI **Dopamine** neurons heterozygous for the Nurr1-null allele have reduced survival in vitro

L6 ANSWER 14 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI Will embryonic stem cells be a useful source of **dopamine** neurons for transplant into patients with Parkinson's disease?

L6 ANSWER 15 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI Protective effect of insulin-like-**growth**-factor-1 against

TI Chronic levodopa impairs morphological development of grafted embryonic **dopamine** neurons

L9 ANSWER 7 OF 44 CA COPYRIGHT 2005 ACS on STN

TI Differential coupling with pertussis toxin-sensitive G proteins of **dopamine** and somatostatin receptors involved in regulation of adenohipophyseal secretion

L9 ANSWER 8 OF 44 CA COPYRIGHT 2005 ACS on STN

TI Normal and adenomatous human pituitaries secrete thyrotropin-releasing hormone in vitro: modulation by **dopamine**, haloperidol, and somatostatin

L9 ANSWER 9 OF 44 CA COPYRIGHT 2005 ACS on STN

TI Long-term **culture** of rat mammothroph and somatotroph subpopulations separated on continuous Percoll density gradients: effects of **dopamine**, TRH, GHRH and somatostatin

L9 ANSWER 10 OF 44 CA COPYRIGHT 2005 ACS on STN

TI A muscle-derived factor(s) induces expression of a **catecholamine** phenotype in neurons of cultured rat cerebral cortex

L9 ANSWER 11 OF 44 CA COPYRIGHT 2005 ACS on STN

TI The effect of **epinephrine** and benzalkonium chloride on cultured corneal endothelial and trabecular meshwork cells

L9 ANSWER 12 OF 44 CA COPYRIGHT 2005 ACS on STN

TI Dihydropyridine-sensitive calcium channel activity related to prolactin, **growth** hormone, and luteinizing hormone release from anterior pituitary cells in **culture**: interactions with somatostatin, **dopamine**, and estrogens

L9 ANSWER 13 OF 44 CA COPYRIGHT 2005 ACS on STN

TI Enhancement of the response of hen granulosa cells to LH with **norepinephrine** in vitro

L9 ANSWER 14 OF 44 CA COPYRIGHT 2005 ACS on STN

TI Evidence that prostaglandins activate calcium channels to **enhance** basal and stimulation-evoked **catecholamine** release from bovine adrenal chromaffin cells in **culture**

L9 ANSWER 15 OF 44 CA COPYRIGHT 2005 ACS on STN

TI Survival and function of dissociated rat **dopamine** neurons grafted at different developmental stages or after being cultured in vitro

L9 ANSWER 16 OF 44 CA COPYRIGHT 2005 ACS on STN

TI Modification by pertussis toxin of the responses of bovine anterior pituitary cells to acetylcholine and **dopamine**: effects on hormone secretion and rubidium-86 efflux

L9 ANSWER 17 OF 44 CA COPYRIGHT 2005 ACS on STN

TI Effects of retinoic acid on alkaline phosphatase messenger ribonucleic acid, **catecholamine** receptors, and G proteins in ROS 17/2.8 cells

L9 ANSWER 18 OF 44 CA COPYRIGHT 2005 ACS on STN

TI Survival, morphology, and **catecholamine** storage of chromaffin cells in serum-free **culture**: evidence for a survival and differentiation promoting activity in medium conditioned by purified chromaffin cells

L9 ANSWER 19 OF 44 CA COPYRIGHT 2005 ACS on STN

TI Differential effects of nerve **growth** factor and ciliary neuronotrophic factor on **catecholamine** storage and

**catecholamine** synthesizing enzymes of cultured rat chromaffin cells

- L9 ANSWER 20 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Evidence that stimulation of **growth** hormone release by **epinephrine** and vasoactive intestinal peptide is based on **cell-to-cell** communication in the pituitary
- L9 ANSWER 21 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI  $\alpha$ 2-Adrenoceptors do not regulate **catecholamine** secretion by bovine adrenal medullary cells: a study with clonidine
- L9 ANSWER 22 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Role of **cell-to-cell** communication in the **growth** hormone response to **epinephrine**, **growth** hormone releasing factor and vasoactive intestinal peptide
- L9 ANSWER 23 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI **Catecholamine** modulation of embryonic palate mesenchymal **cell** DNA synthesis
- L9 ANSWER 24 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Relationship between **dopamine** content and its secretion in PC12 cells as a function of **cell growth**
- L9 ANSWER 25 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Effects of **growth** hormone-releasing factor(1-44) on **growth** hormone release from human somatotropinomas in vitro: interaction with somatostatin, **dopamine**, vasoactive intestinal peptide and cycloheximide
- L9 ANSWER 26 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Effect of extracellular matrix on PC 12 **cell** shape and **dopamine** processing
- L9 ANSWER 27 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Inhibition of baboon marrow CFU-GEMM, CFU-GM, BFU-E and CFU-E by adrenochrome, an **epinephrine** metabolite
- L9 ANSWER 28 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Hypothyroid pituitary cells in **culture**: an analysis of thyrotropin and prolactin responses to **dopamine** (DA) and DA receptor binding
- L9 ANSWER 29 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Effects of ascorbic acid, dexamethasone, and insulin on the **catecholamine** and opioid peptide stores of cultured adrenal medullary chromaffin cells
- L9 ANSWER 30 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Presence of nerve **growth** factor receptors and **catecholamine** uptake in subpopulations of chick sympathetic neurons: correlation with survival factor requirements in **culture**
- L9 ANSWER 31 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Angiotensin II stimulates changes in the **norepinephrine** content of primary cultures of rat brain
- L9 ANSWER 32 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Suppression of **catecholamine** and melanin synthesis and promotion of cholinergic differentiation of quail neural crest cells by heart **cell** conditioned medium
- L9 ANSWER 33 OF 44 CA COPYRIGHT 2005 ACS on STN

L13 ANSWER 1 OF 10 TOXCENTER COPYRIGHT 2005 ACS on STN DUPLICATE 1

TI An *in vitro* bacterial model of cytotoxicity to living cells caused by **dopamine** and 6-hydroxydopamine oxidation at physiological pH

AN 1991:143080 TOXCENTER

CP Copyright 2005 ACS

DN CA11503023921H

TI An *in vitro* bacterial model of cytotoxicity to living cells caused by **dopamine** and 6-hydroxydopamine oxidation at physiological pH

AU Giunta, Sergio; Galeazzi, Luciano; Groppa, Giuseppe

CS Ist. Carattere Sci., INRCA, Rome, Italy.

SO Free Radical Biology & Medicine, (1991) Vol. 10, No. 5, pp. 297-303.

CODEN: FRBMEH. ISSN: 0891-5849.

CY ITALY

DT Journal

FS CAPLUS

OS CAPLUS 1991:423921

LA English

ED Entered STN: 20011116

Last Updated on STN: 20021008

TI An *in vitro* bacterial model of cytotoxicity to living cells caused by **dopamine** and 6-hydroxydopamine oxidation at physiological pH

SO Free Radical Biology & Medicine, (1991) Vol. 10, No. 5, pp. 297-303.

CODEN: FRBMEH. ISSN: 0891-5849.

AB The cytotoxicity of dopamine (DA) and 6-hydroxydopamine (6-OHDA) on living cells, *in vitro*, has been previously investigated in neuroblastoma cells. This study was designed to explore the possibility of using **bacteria** as targets for studying DA and 6-HODA cytotoxicity. Both DA and 6-HODA oxidize when added to bacteriol. media. The rate of autoxidn. of 6-HODA was greater than DA within the first hours. The oxidation-dependent cytotoxicity caused bacterial **growth** -inhibition and killing at concentration of 10-4M. All the bacterial strains tested were slightly more susceptible to DA than to 6-HODA. Antioxidants (sodium metabisulfite, cysteine) prevented the oxidation and abolished the **growth**-inhibitory activity. The addition of exogenous catalase protected the cells against the effect of the oxidation of both the catecholamines up to the concentration of 5 mM, while the addition of exogenous superoxide dismutase protected the cells only at the minimal inhibitory concns. Taking into account that some of the results obtained are similar to those previously reported using neuroblastoma cells as targets, the use of **bacteria** for studying oxygen toxicity from these catecholamines seems to be a potentially useful model system.

ST Miscellaneous Descriptors

dopamine oxidn cytotoxicity **bacteria** oxygen radical;

hydroxydopamine oxidn cytotoxicity **bacteria** oxygen radical

AB The cytotoxicity of dopamine (DA) and 6-hydroxydopamine (6-OHDA) on living cells, *in vitro*, has been previously investigated in neuroblastoma cells. This study was designed to explore the possibility of using **bacteria** as targets for studying DA and 6-HODA cytotoxicity. Both DA and 6-HODA oxidize when added to bacteriol. media. The rate of autoxidn. of 6-HODA was greater than DA within the first hours. The oxidation-dependent cytotoxicity caused bacterial **growth** -inhibition and killing at concentration of 10-4M. All the bacterial strains tested were slightly more susceptible to DA than to 6-HODA. Antioxidants (sodium metabisulfite, cysteine) prevented the oxidation and abolished the **growth**-inhibitory activity. The addition of exogenous catalase protected the cells against the effect of the oxidation of both the catecholamines up to the concentration of 5 mM, while the addition of exogenous superoxide dismutase protected the cells only at the minimal inhibitory

concns. Taking into account that some of the results obtained are similar to those previously reported using neuroblastoma cells as targets, the use of **bacteria** for studying oxygen toxicity from these catecholamines seems to be a potentially useful model system.

L13 ANSWER 2 OF 10 TOXCENTER COPYRIGHT 2005 ACS on STN DUPLICATE 2  
TI Inhibition of human immunodeficiency **virus** type 1 replication and cytopathicity by synthetic soluble **catecholamine** melanins in **vitro**  
AN 1990:33630 TOXCENTER  
DN PubMed ID: 2327999  
TI Inhibition of human immunodeficiency **virus** type 1 replication and cytopathicity by synthetic soluble **catecholamine** melanins in **vitro**  
AU Montefiori D C; Modliszewski A; Shaff D I; Zhou J  
CS Department of Pathology, Vanderbilt University Medical School, Nashville, Tennessee 37232  
SO Biochemical and biophysical research communications, (1990 Apr 16 ) 168 (1) 200-5.  
Journal Code: 0372516. ISSN: 0006-291X.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
FS MEDLINE  
OS MEDLINE 90226360  
LA English  
ED Entered STN: 20011116  
Last Updated on STN: 20011116  
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SO Biochemical and biophysical research communications, (1990 Apr 16 ) 168 (1) 200-5.  
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AB Synthetic soluble melanins were synthesized by spontaneous oxidation of L-dopamine, norepinephrine or 5-hydroxytryptamine (serotonin) in weak alkaline solution. These three melanins inhibited infection of human CD4+ lymphoblastoid cells (MT-2) by cell-free human immunodeficiency **virus** type 1 (HIV-1), without cell toxicity, at concentrations of 0.15-10 micrograms/ml. Also, syncytium formation and resulting cytopathic effects when uninfected cells were mixed with chronic HIV-1-infected cells were blocked by these melanins. Antisyncytial activity was greater when infected cells were preincubated with melanin than when uninfected cells were preincubated with melanin, thus suggesting that interaction of melanin with viral proteins is an important aspect of the antiviral mechanism. These results make synthetic soluble melanins interesting candidates for further study as possible anti-HIV-1 therapeutics.  
CT Check Tags: In Vitro  
\*Catecholamines: PD, pharmacology  
Cell Fusion: DE, drug effects  
Cytopathogenic Effect, Viral: DE, drug effects  
HIV-1: DE, drug effects  
\*HIV-1: GD, growth & development  
Humans  
\*Melanins: PD, pharmacology  
\***Virus** Replication: DE, drug effects  
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L9 ANSWER 28 OF 44 CA COPYRIGHT 2005 ACS on STN

TI Hypothyroid pituitary cells in **culture**: an analysis of thyrotropin and prolactin responses to **dopamine** (DA) and DA receptor binding

AN 101:66401 CA

TI Hypothyroid pituitary cells in **culture**: an analysis of thyrotropin and prolactin responses to **dopamine** (DA) and DA receptor binding

AU Foord, Steven M.; Peters, John R.; Dieguez, Carlos; Jasani, Barhat; Hall, Reginald; Scanlon, Maurice F.

CS Dep. Med., Welsh Natl. Sch. Med., Heath Park/Cardiff, CF4 4XN, UK

SO Endocrinology (1984), 115(1), 407-15

CODEN: ENDOAO; ISSN: 0013-7227

DT Journal

LA English

TI Hypothyroid pituitary cells in **culture**: an analysis of thyrotropin and prolactin responses to **dopamine** (DA) and DA receptor binding

SO Endocrinology (1984), 115(1), 407-15

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AB After 3 days in **culture**, the anterior pituitary (AP) cells from hypothyroid rats showed greater TSH [9002-71-5] and prolactin (PRL) [9002-62-4] secretory activity and less **growth** hormone (GH) [9002-72-6] secretory activity than did parallel euthyroid cultures. Bromocriptine [25614-03-3], apomorphine [58-00-4], and dopamine (DA) [51-61-6] inhibited euthyroid TSH secretion by .apprx.30%, whereas each drug inhibited hypothyroid TSH secretion by .apprx.60%. In contrast, the 3 agonists were less effective in inhibiting PRL secretion from hypothyroid cells. The rank order of potency [bromocriptine > (+)butaclamol [56245-67-1] > apomorphine > DA > (-)butaclamol [51152-91-1] shown against secretion was the same for TSH and PRL in both euthyroid and hypothyroid **cell** cultures and is typical of a DA receptor-mediated process. The binding of [3H]dihydroergocryptine (DHE) to DA receptors on euthyroid and hypothyroid cells was examined. One micromolar concentration of (+)butaclamol was used to define nonspecific binding.

Specific

binding was saturable and stereospecific in each case. The rank order of potency of dopaminergic agonists and antagonists in competing for [3H]DHE binding was the same as that demonstrated against the secretion of TSH and PRL. Each compound displayed more [3H]DHE from hypothyroid cells than from euthyroid cells. Construction of adsorption isotherms for [3H]DHE binding DA receptors on euthyroid and hypothyroid cells and subsequent Scatchard anal. revealed a 3-4-fold increase in receptor number without a change in affinity. Immunohistochem. on AP lobes before and after dispersion revealed an increase in thyrotrophs and thyroidectomy cells in hypothyroid rats relative to those in control animals. In euthyroid animals thyrotrophs were 10.1% of the total AP **cell** population, in hypothyroid animals thyrotrophs plus the thyroidectomy cells were 36.3% of the total AP cells. Therefore, the increased number of DA receptors per lobe could be accounted for by increased nos. of thyrotrophs. The mechanism of the altered sensitivity to DA induced by hypothyroidism in lactotrophs and thyrotrophs remains to be clarified.

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(FILE 'HOME' ENTERED AT 13:22:16 ON 12 SEP 2005)

FILE 'CAPLUS' ENTERED AT 13:22:39 ON 12 SEP 2005  
L1 0 S DYER/AU AND VOL AND 102 OR MOGER/AU AND VOL 97 AND 1982  
L2 0 S DYER AND VOL AND 102 OR MOGER AND VOL 97 AND 1982  
L3 0 S DYER.INV AND VOL AND 102 OR MOGER.INV AND VOL 97 AND 1982  
E DYER

FILE 'CA' ENTERED AT 13:26:08 ON 12 SEP 2005  
L4 0 S (DYER/INV OR DYER/AU) AND VOL 102 AND 1985  
E DYER  
L5 490 S (GROWTH OR ENHANCE OR ENHANCING) AND (CATECHOLAMINE OR EPINEP  
L6 105 S (GROWTH OR ENHANCE OR ENHANCING) AND (CATECHOLAMINE OR EPINEP  
L7 0 S L6 AND @PY<1991  
L8 0 S L6 AND @PY=<1991  
L9 44 S L6 AND PY=<1991